Journal of Traumatic Stress, Vol. 16, No. 6, December 2003, pp. 535–543 (© 2003)

Causes of Death Among Male Veterans Who Received Residential Treatment for PTSD

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Previous studies have shown elevated mortality among psychiatric and substance abusing patients, including veterans with PTSD. Although early studies showed elevated deaths from external causes among Vietnam veterans in the early postwar years, more recent studies have also shown increased health problems among veterans with PTSD. This study compared mortality due to behavioral causes versus other diseases among 1,866 male veterans treated for PTSD. Death certificates obtained for 110 veterans indicated behavioral causes accounted for 62.4% of deaths, standardized mortality ratio = 3.4–5.5, including accidents (29.4%), chronic substance abuse (14.7%), and intentional death by suicide, homicide, or police (13.8%). Results suggest possible opportunities to improve outcomes of this at-risk patient population through harm reduction interventions and improved continuity of care.

KEY WORDS: posttraumatic stress; military veterans; mortality; PTSD treatment.

Research (Kasprow & Rosenheck, 2000) suggests that veterans with PTSD continue to be at risk for premature mortality many years after the end of their military service. This is consistent with well-known data demonstrating increased mortality among psychiatric patients (Felker, Yazel, & Short, 1996), and with research showing increased mortality associated with alcohol and drug use (Dawson, 2000; Kallan, 1998; Shaper, 1990), and depression (Wulsin, Vaillant, & Wells, 1999).

However, the specific causes of premature deaths among Vietnam veterans with PTSD are unclear. Such early deaths may be due to external causes, substance use, and/or other disease processes. Military veterans being treated for chronic PTSD frequently present with other serious risk behaviors and multiple comorbid medical and psychological conditions. For example, impulsivity, aggression, depression, and substance abuse are common problems among veterans with PTSD that could increase their risk of sudden death from external causes such as accidents, suicides, and homicides (Begic & Jokic-Begic, 2001; Kotler, Iancu, Efroni, & Amir, 2001; McFall, Fontana, Raskind, & Rosenheck, 1999). Chronic substance abuse among veterans with PTSD could also contribute to early death directly (e.g., liver disease) or through increased exposure to infectious diseases like HIV and hepatitis C (Bullman & Kang, 1994; Hoff, Beam-Goulet, & Rosenheck, 1997). As they age, Vietnam veterans with PTSD might also face increased mortality risks from heart disease, cancer, and other medical conditions related to high rates of smoking, deficits in self-care, poor medical compliance, and the physiological correlates of chronic stress, hostility, and depression.

Pre-1990 Studies of Mortality Among Vietnam Veterans

After the Vietnam war, as concerns for the fate of returning veterans began to be voiced, a number of studies

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emerged comparing mortality rates and causes of death for Vietnam veterans to those among Vietnam-era veterans deployed in other theaters (Anderson et al., 1986; Breslin, Kang, Lee, Burt, & Shepard, 1988; Centers for Disease Control Vietnam Experience Survey [CDC], 1987; Holmes et al., 1986; Kogan & Clapp, 1985; Lawrence et al., 1985; Watanabe & Kang, 1996). However, only one of these studies examined deaths beyond 1985, extending through 1990. Several studies found increased deaths due to external causes among Vietnam veterans compared to other Vietnam-era veterans and to community controls. Six of seven studies found increased deaths due to motor vehicle accidents, five found increased accidental deaths other than motor vehicle accidents (including accidental overdoses), three found increased suicides, and three found increased homicides. Regarding elevated deaths due to common medical conditions, four studies found increased cancer deaths, although the forms of cancer were not consistent across studies. No studies reported elevated deaths due to heart disease. The study by the CDC (1987) found that excess mortality of Vietnam veterans occurred primarily in the first 5 years after military discharge.

To date, only one study has considered causes of death specifically among veterans with PTSD. Bullman and Kang (1994) studied male Vietnam veterans entered into the Agent Orange Registry between 1982 and 1990 with and without a PTSD diagnosis. The PTSD group had a 71% higher overall mortality rate than the non-PTSD veterans group, including higher rates of death from all accidents (and accidental overdose in particular) and from suicide. Veterans in the PTSD group were also more likely to die of diseases of the digestive system (primarily cirrhosis of the liver) than were age- and ethnicity-matched controls from the general population.

Post-1990 Mortality Among Vietnam Veterans with PTSD

To date there is little information on patterns of mortality among veterans with chronic PTSD over a broader lifespan. Deaths from external causes might decrease in later years, due to selection (i.e., those veterans most likely to die from accidents, suicide, or violence may have already died) and the passage of time since their service in the combat zone. Deaths related to chronic substance abuse might increase over time because of cumulative damage and more opportunity for exposure to diseases like HIV.

Aging combat veterans with PTSD might also be at elevated risk for deaths due to medical causes such as heart disease or cancer. For example, smoking has been found to be elevated among veterans with PTSD (Beckham et al., 1997), and combat-related PTSD has been associated with increased health problems even after controlling for alcohol use and smoking (Schnurr & Jankowski, 1999). Psychological factors such as depression and hostility, that frequently co-occur with PTSD, present known increased health risks for cardiovascular disease (Dwight & Stoudemire, 1997; Goldstein & Niaura, 1995; Musselman, Evans, & Nemeroff, 1998; Musselman & Nemeroff, 2000). Depression is also associated with compromised immune function (Connor & Leonard, 1998), and poor medical compliance (DiMatteo, Lepper, & Croghan, 2000).

A recent study (Kasprow & Rosenheck, 2000) found that male veterans receiving outpatient treatment for PTSD in 1989 or 1990 had a significantly higher risk of death prior to 1999 than did U.S. males in the general population (SMR = 3.2). Although this study found that veterans seeking treatment for chronic PTSD continue to be at risk for early death during middle age, it presented no information on the causes of those excess deaths. It is therefore unclear to what extent these deaths are due to behavioral causes (external causes and direct effects of substance use) or other disease processes (including stress-related illness). If a significant proportion of those deaths stem from behavioral causes, treatment efforts should be expanded to include reducing related risk behaviors.

It is currently unclear whether high rates of deaths from external causes identified in the early studies of mortality among Vietnam veterans have continued. It is also unclear at present whether deaths expected to occur from increase stress-related health risks are becoming apparent among those veterans with chronic war-related PTSD as they age. Accordingly, this study will first examine death rates and causes of death among veterans admitted for residential PTSD treatment. Secondarily, we will focus on the relative contribution of behavioral deaths and other disease processes to the causes of deaths within this population.

Method

Sample

The cohort for this study was consecutive male admissions to a VA PTSD residential rehabilitation treatment program ($N = 1{,}866$) that occurred between January 1, 1990 and December 31, 1998. Admission to this program is limited to clinician-referred veterans with

military-related traumatic exposure and PTSD. Military and overseas service was confirmed at time of admission by review of the veteran's discharge record (DD-214). All individuals were substance free for at least 15–30 days prior to admission. Individuals with active psychosis, current legal problems, or severe medical conditions were excluded from admission. The consecutive patient list was generated from program database records actively maintained since 1990. PTSD treatment was provided in group format with groups primarily utilizing cognitive-behavioral skills training, and interpersonal process approaches. Patients deemed clinically appropriate also participated in trauma focus groups where specific military and combat-related trauma were processed. Information about patients with multiple admissions to this program during these years (6.4% of sample) was taken from the last admission. In addition to PTSD, other comorbid diagnoses were highly prevalent within this sample. Diagnostic interviews were conducted with most patients (N = 1,682) upon admission utilizing the Structured Clinical Interview for DSM-IIIR and DSM-IV (SCID). Results indicated that 81.2% met SCID criteria for history of major depression, 4.6% for history of bipolar disorder, 67.2% for history of alcohol dependence, and 49.9% for history of drug dependence. Demographic characteristics of this sample can be seen in Table 1.

Table 1. Demographic Characteristics for PTSD Residential Rehabilitation Patients (N = 1,866)

	%	*M*	*SD*
Age at treatment discharge		47.18	5.07
Education		13.33	2.02
Ethnicity			
Caucasian	66.2		
African American	11.8		
Hispanic	13.4		
Native American	3.7		
Other	4.9		
Marital Status			
Never married	8.5		
Married	31.8		
Live w/partner	3.2		
Divorced	27.4		
Separated	27.7		
Widowed	1.3		
Branch of service			
Army	65.3		
Navy	7.1		
Air Force	3.3		
Marine	24.3		
Vietnam theater	95.7		
Length of stay (days)		89.68	49.23
Years to follow-up		5.00	2.56

Note. Missing data: ethnicity (n = 14), education (n = 635), marital status (n = 603), branch (n = 617), and Vietnam theater (n = 636).

Determination of Mortality

The authors examined mortality status and patterns among patients who were identified as deceased prior to December 31, 1999 by an internet search of the Social Security Death Index (SSDI, updated after December 31, 1999). Both Hill (2001) and Sesso, Paffenbarger, and Lee (2000) suggest that the use of web-based searches of the Social Security Death Master File is an "alternative, inexpensive method of determining the mortality status of subjects in relatively small epidemiologic studies," particularly when subjects are males. In situations (including this study) where correct Social Security Numbers are available, the SSDI yields only slightly fewer correct matches (83.6% vs. 93.1%) while returning fewer false matches (0.5% vs. 10.4%, Curb et al., 1985) than the National Death Index (NDI). After identifying the deceased individuals, a request for cause of death information was sent to NDI. Coded cause of death information was obtained for 99% of the 110 (109 of 110) deceased individuals identified by the SSDI search.

Definition of Behavioral Causes of Death

This study compared the prevalence of deaths due to behavioral causes and deaths due to other disease processes. Behavioral deaths were intended to include deaths associated with high-risk behaviors that are potentially amenable to psychosocial intervention. This includes deaths from external causes (both accidental and intentional deaths), and from causes directly linked to substance use (alcoholic cirrhosis, HIV/hepatitis, and other deaths where substance use was listed as a primary cause of death). These were contrasted with deaths from heart disease, cancer, and other diseases.

Causes of death were combined into seven major categories, on the basis of ICD-9 causes of death category codes. Four categories were included in our definition of behavioral causes: (1) accidents, including motor vehicle accidents, accidental overdoses of drugs or alcohol, other accidents such as fires, or falls, and injury deaths of undetermined intent; (2) intentional deaths, including suicides, homicides, and individuals killed in encounters with the police; (3) chronic effects of substance use, including deaths from alcoholic liver cirrhosis and death certificates where alcohol or drug dependence is listed as the primary cause of death; and (4) HIV or hepatitis (infectious diseases associated with high risk IV drug use or sexual behavior). We also conducted a secondary analysis of overall deaths related to substance use, combining deaths from accidental overdoses (from the accidents category) with deaths from chronic substance use. Three categories

included other disease processes, specifically (5) heart disease; (6) cancer; and (7) all other causes of death. Notably, cancers caused by smoking were not included as behavioral deaths. Although these deaths would fit the definition conceptually, there is no ICD-9 code in mortality records that would enable the authors to determine which deaths were likely to be smoking related. Analysis of behavioral deaths also did not include cases where poor diet, lack of exercise, lack of medical care due to isolation and distrust, or poor medical compliance may have contributed to medical mortality.

Data Analysis

Comparing mortality due to external and behavioral causes (intentional deaths, accidents, chronic substance use, and infections related to high-risk behavior) and other disease processes (heart disease, cancer, and other causes) requires adjustment for the rates of mortality due to these causes in the general population. The authors therefore expressed mortality risks in terms of standardized mortality ratios (SMRs, i.e., the observed mortality divided by the expected mortality rate).

Expected mortality rates were derived from general population norms using simultaneous direct adjustment standardization (Fleiss, 1981). We simultaneously stratified the sample on three dimensions: age at discharge (5-year increments), race (White vs. non-White), and number of years followed (1–9 years). Expected mortality rates (from all causes) within each stratum were determined using data from "Abridged life tables for white males, 1996" and "Abridged life tables for all other males, 1996" (National Center for Health Statistics [NCHS], 1998). Mortality rates in each stratum were weighted by the distribution of these strata in the sample and summed to generate an overall expected mortality rate. Although mortality risks in the general population vary slightly from year to year, such differences would have only minimal effects on expected values in a small population of under 2,000 patients, and therefore used 1996 data for overall risk and 1997 data for distribution of causes of death among those who died.

For example, a stratum of 40-year-old non-White males followed for 3 years, would have expected mortality determined as follows: Expected mortality = 1 − [(survival rate from age 40 to 41) × (survival rate from age 41 to 42) × (survival rate from age 42 to 43)]. In this case, expected 3-year mortality rate for this stratum would be 1 − (.9948 × .9950 × .9941) = .0159. We then multiplied expected mortality for each age-race-years followed stratum by the number of subjects in our sample who were in each stratum to generate a weighted estimate of overall expected mortality.

Determining expected mortality due to a specific cause, such as chronic substance use, involved an additional step. For the first year followed, the 1-year mortality rate for each stratum in the general population (NCHS, 1998) was multiplied by the proportion of deaths in that stratum in the general population that were due to the particular cause (NCHS, 2000). This process was repeated for each year followed, using conditional probability to sum the expected likelihood of survival, death due to a given cause, and death due to other causes across the total number of years followed for each age and race stratum. These analyses were repeated for each cause of death reported in this study. Cause-specific expected mortality rates in each stratum were again weighted by the distribution of strata in our sample to generate overall expected mortality rates due to each cause.

For example, we estimated a 41-year-old non-White man's likelihood of dying from chronic substance use within 1 year as the 1-year mortality rate from age 40 to age 41 (.0052) times the proportion of all deaths between ages 40 and 44 that were due to chronic substance use (.033), or (.0052 × .033) = .00017. Conditional probability was used to calculate cumulative risk of dying from each specific cause over multiple years. For example, expected 3-year mortality due to chronic substance use = (probability of dying from substance use in year 1) + (probability of surviving year 1 times probability of dying from substance use in year 2) + (probability of surviving years 1 and 2 times probability of dying from substance use in year 3). We then multiplied expected cumulative mortality due to chronic substance use for each stratum by the number of subjects in our sample who were in each stratum to generate a weighted estimate of overall expected mortality due to chronic substance use.

Both overall mortality and specific-cause mortality were analyzed using standardized mortality ratios (observed number of deaths/expected number of deaths), using Kahn and Sempos's procedure for calculating confidence intervals (CIs, Sempos, 1989). To control for experiment-wise error, causes of death were analyzed in three hierarchical steps. First, the overall pattern of observed causes of death in our clinical sample was compared to expected values for the general population. If this overall analysis proved significant (i.e., the entire confidence interval was greater than 1), follow-up analyses were conducted comparing the observed and expected numbers of deaths due to each major category. Finally, if any major category (e.g., accidents) was a significantly more common cause of death than would be expected in the general population, further analyses were conducted to identify which specific subcategories (e.g., motor vehicle accidents, accidental overdoses, other

accidents) contributed higher-than-expected numbers of deaths.

Results

Mortality

Of the entire sample of 1,866 PTSD patients followed for 1–9 years, 110 (5.9%) died during the study period. This corresponds to an average mortality of roughly 1.2% per year. Total mortality was higher than would be expected in a general population sample with a similar age and race distribution followed for the same length of time (SMR = 1.8, 95% CI = 1.5–2.2).

Causes of Death

The numbers of deaths were calculated for the major groupings of deaths identified as associated with behavioral causes and for other medical conditions (heart disease, cancer, and all other causes). Table 2 shows those percentages for the total sample as well as for Whites and non-Whites.

It is important to note that 62.4% of all deaths in these PTSD patients occurred from what was termed behavioral causes. Over one third (37.6%) of all deaths were directly attributable to either acute or chronic effects of alcohol or drug use. There was some indication of ethnic differences in the accidents category, $\chi^2(1, N = 109) = 3.9$, $p < .05$, with non-Whites having higher rates of accidents, primarily overdoses.

Figure 1 shows the actual number of deaths in the PTSD group compared to the number of deaths that would be expected to occur in the general population based on CDC mortality tables adjusted for age and race.

Standardized mortality ratios for behavioral causes (combining accidents, intentional deaths, effects of chronic substance use, and HIV/hepatitis) were highly elevated, SMR = 4.4, (95% CI = 3.4–5.5). In contrast, deaths from other disease processes (combining cancer, heart disease, and other causes) were not higher than expected in the general population, SMR = 0.9 (95% CI = 0.7, 1.2).

Table 2. Causes of Death for PTSD Residential Rehabilitation Patients ($N = 109$)

Cause	Total PTSD		White		Non-White	
	N	%	*N*	%	*N*	%
Deaths due to behavioral causes	68	62.4	47	61.8	21	63.6
Accidents	32	29.4	18	23.7	14	42.4
Motor vehicle accident	10	9.2	7	9.2	3	9.1
Accidental overdose	13	11.9	7	9.2	6	18.2
Other accidents	6	5.5	3	3.9	3	9.1
Unknown intent	3	2.8	1	1.3	2	6.1
Injury	1	0.9	0	0.0	1	3.0
Alcohol/drug overdose	2	1.8	1	1.3	1	3.0
Intentional deaths	15	13.8	12	15.8	3	9.1
Suicide	9	8.3	7	9.2	2	6.1
Firearms	5	4.6	4	5.3	1	3.0
Alcohol/drug overdose	2	1.8	2	2.6	0	0.0
Other suicides	2	1.8	1	1.3	1	3.0
Homicide	4	3.7	3	3.9	1	3.0
Firearms	2	1.8	2	2.6	0	0.0
Other homicides	2	1.8	1	1.3	1	3.0
Killed by police	2	1.8	2	2.6	0	0.0
Chronic substance use	16	14.7	13	17.1	3	9.1
Liver disease—alcohol related	8	7.3	6	7.9	2	6.1
Alcohol/drug dependence	8	7.3	7	9.2	1	3.0
High-risk IV drug/sexuality	5	4.6	4	5.3	1	3.0
HIV	3	2.8	3	3.9	0	0.0
Viral hepatitis	2	1.8	1	1.3	1	3.0
Nonbehavioral deaths	38	34.9	27	35.5	11	33.3
Cancer	7	6.4	3	3.9	4	12.1
Heart disease	14	12.8	12	15.8	2	6.1
Other diseases	17	11.0	12	6.6	5	15.2
Unknown cause	3	2.8	2	2.6	1	3.0
Total	109	100.0	76	100.0	33	100.0
Deaths with alcohol/drug abuse as secondary cause	3	2.8	1	1.3	2	6.1
Total substance-related deaths	41	37.6	28	36.8	13	39.4

Note. $N = 109$ because cause of death information from NDI was not obtainable for one participant.

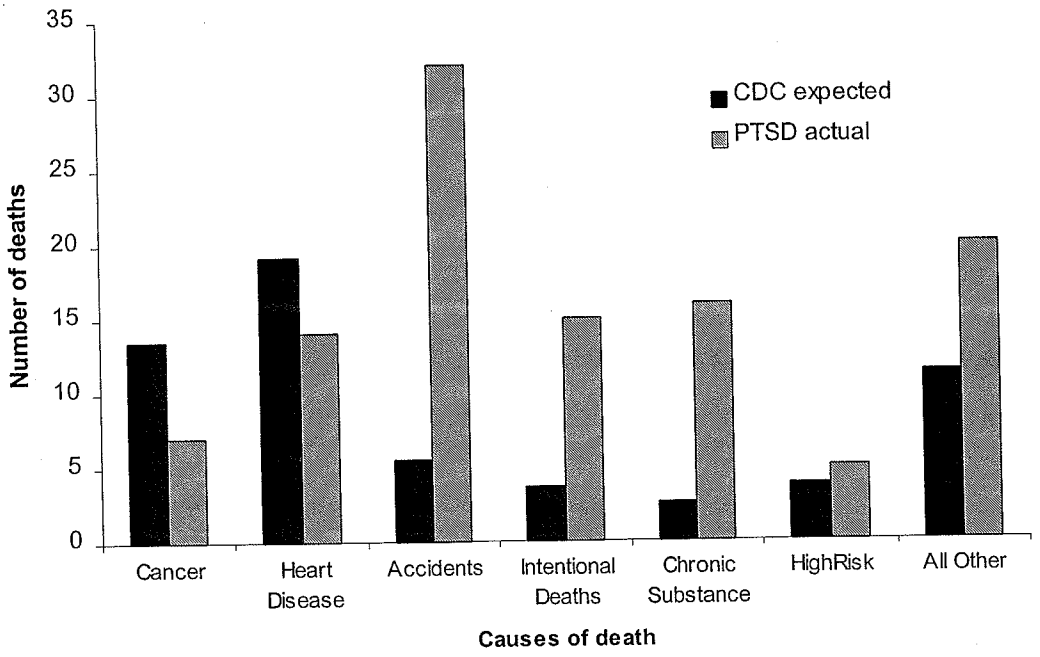

Fig. 1. Actual and expected causes of PTSD death, adjusted for age and race ($N = 109$).

Looking at more specific causes, there were significantly increased rates of death for accidents, SMR = 5.7 (95% CI = 3.9–8.1), and for the sub-categories motor vehicle accidents, SMR = 4.4 (95% CI = 2.1–8.0), accidental overdose, SMR = 9.6 (95% CI = 5.1–16.5), injury undetermined whether accidentally or purposely inflicted, SMR = 7.8 (95% CI = 1.6–22.7), and other accidents, SMR = 4.6 (95% CI = 1.7–10.0). PTSD patients also had a higher-than-expected number of intentional deaths, SMR = 4.1 (95% CI = 2.3–6.8). Within this category, there were significantly elevated deaths due to suicide, SMR = 4.0 (95% CI = 1.8–7.4), but not for homicide, SMR = 2.9 (95% CI = 0.8–7.4). The SMR for death by police was elevated, 80.9 (95% CI = 10.1–301.0), but should be interpreted with caution as SMR's may be unreliable for events with extremely low base-rates. Previous studies (Kogan & Clapp, 1985) have also examined a category called "estimated suicide rate," where suicide deaths are combined with all overdose deaths and deaths of unknown cause. When these categories are combined the result for the PTSD group is significantly higher than expected death rates within the general population, SMR = 5.6 (95% CI = 3.7–8.1).

Elevated deaths were observed for sequelae of chronic substance use, SMR = 6.1 (95% CI = 3.5–9.9), and for the sub-categories alcoholic liver cirrhosis, SMR = 4.8 (95% CI = 2.1–9.5) and alcohol dependence, SMR = 8.4 (95% CI = 3.6–16.5). Deaths from infectious diseases related to IV drug use and high-risk sexual behavior were not significantly elevated among PTSD patients, SMR = 1.3 (95% CI = 0.4–3.1). Deaths from liver disease without mention of alcohol, although not included in this category (it is included in all other diseases), was also significantly higher in the PTSD group, SMR = 7.2 (95% CI = 2.8–14.6).

Finally, the proportion of deaths in the PTSD group from the two most frequent causes of death among males of similar age in the general population, cancer, SMR = 0.5 (95% CI = 0.2–1.1), and heart disease, SMR = 0.7 (95% CI = 0.4–1.2), were not significantly different than expected.

Discussion

These data indicate several findings with clinical relevance for veterans in treatment for PTSD. First, results are consistent with the finding by Kasprow and Rosenheck (2000) that the death rate for veterans seeking treatment for PTSD in recent years is higher than the rate for the general population of comparable males. This is also consistent with other studies showing elevated mortality among psychiatric patients (Dawson, 2000; Felker et al., 1996; Kallan, 1998; Shaper, 1990; Wulsin et al., 1999).

Second, this study analyzed which specific causes contributed to this elevated mortality among middle-aged

Vietnam veterans with PTSD. Nearly two thirds of deaths in this study were attributable to potentially preventable behavioral causes, including accidents, intentional deaths, and causes related to substance abuse and HIV/hepatitis. Moreover, for all but the last of these categories, mortality rates were significantly higher than would be expected in the general population. The fact that such a high proportion of deaths among patients is related to behavioral causes suggests that important treatment opportunities continue to exist to target high-risk behavior patterns for more intensive efforts at reduction during residential treatment for PTSD and afterwards during continuing care.

The finding that deaths from heart disease and cancer did not differ significantly from what would be expected in the general population may appear paradoxical in light of other research indicating that PTSD patients are at elevated risk for these health problems (Schnurr & Jankowski, 1999; Schnurr & Spiro, 1999). It is possible that elevations in deaths due to these causes may only manifest later in time as these veterans continue to age. Also, some veterans who had serious medical conditions that could have led to early death may have actually died first from other external causes. Thus, it remains unclear whether chronic PTSD patients are in fact at the same or lower than average risk of mortality from heart disease or cancer than males in the general population.

These results suggest that the patterns in death rates for external causes seen in the early mortality studies of Vietnam veterans have continued for those veterans with severe PTSD seeking residential treatment. Significant elevations in stress-related disease deaths are not yet evident in this cohort, or are being obscured by the deaths from behavioral causes. Prospective studies using larger, national samples of veterans receiving PTSD treatment are needed to better clarify the mortality risk and patterns associated with chronic combat-related PTSD. Studies that can differentiate the relative contribution to mortality rates of PTSD alone from that associated with comorbid disorders such as depression and substance dependence would also be useful. Additionally, it would be helpful to pursue studies designed to determine whether the higher mortality risk is associated with PTSD itself or shared by VA inpatients generally, by comparing mortality rates across different VA treatment samples and controlling for diagnosis.

These data suggest the need for enhanced clinical attention toward harm reduction strategies designed to reduce morbidity and mortality associated with behavioral causes. Current PTSD treatment programs focus heavily on the reduction of PTSD symptoms and their associated distress. Although this is appropriate for individuals suffering from acute PTSD, it is not sufficient to meet the clinical needs of individuals with more chronic and complex forms of PTSD. Many patients receiving residential PTSD treatment decades after the occurrence of traumatic events have multiple comorbidities and severe chronic life problems, combined with an isolated lifestyle with few resources and supports. The findings suggest that PTSD residential treatment programs could benefit from paying more attention to the current life-threats associated with substance abuse, hostility, violence, and depression.

There has been much discussion of the harm-reduction model in recent years in the substance abuse field. A recent article (MacCoun, 1998) suggests a treatment perspective that utilizes a three-pronged approach to dealing with the harmful consequences of substance use and other risky behaviors. One approach is to discourage people from engaging in the behavior (prevalence reduction), another is to reduce the frequency or extent of the behavior (quantity reduction), and third is to reduce the harmful consequences of the behavior when it occurs (harm reduction). VA treatment programs might do well to address all three approaches in designing more effective interventions for risk behavior patterns associated with chronic combat-related PTSD, and implement them across the entire continuum of care.

Specific targeted interventions should be considered for PTSD subgroups with a clinical history of violent behavior or unsafe weapons ownership and use, DUIs or vehicular accidents, IV-drug use or a history of overdoses, and suicidality. Incorporating these specific high-risk behaviors into the treatment plan, addressing these as part of the clinical intervention process, and providing supportive resources in these areas as part of the discharge plan should become routine aspects of clinical care for these patients. Specific education as to the life and health risks of these high-risk behavior areas, and motivation enhancement interventions (Murphy, Rosen, Cameron, & Thompson, in press) designed to increase veterans' commitments to modify their high risk lifestyles should be considered as part of treatment.

Interventions to reduce risk behaviors associated with mortality are also likely to bring additional benefits in reducing morbidity. For every MVA or accidental overdose resulting in death, there are likely a number of other PTSD veterans hospitalized for these injuries who survive. For every completed suicide, there are likely other PTSD veterans who are hospitalized following a survived suicide attempt. Reductions in such risk behaviors are therefore likely to not only increase veterans' longevity, but also to improve their quality of life. Implementing strategies such as these will require coordinated efforts among those staff responsible for assessment, treatment, discharge planning and continuing care. Sherman et al. (2001) illustrate such a strategy in describing firearms risk

management procedures implemented in an Ohio behavioral health care system.

A primary limitation of this study is that without a comparison group of non-PTSD veterans it is impossible to attribute the findings specifically to PTSD. It may be that other treatment-seeking VA populations (i.e., psychiatric inpatients, homeless, outpatient mental health) might have similar rates and causes of death to that found in this study. However, given that the particular treatment program sampled accepts nearly half of its admissions from diverse areas of the country, it is unlikely that these data reflect simply regional or geographical differences, and are likely to be applicable to patients served by other VA residential PTSD treatment programs throughout the country.

It is important to recognize that veterans treated in VA residential PTSD programs frequently have extremely chronic and severe symptoms and life dysfunction with multiple comorbid disorders, and may differ greatly from patients treated in other settings. Frequently individuals are referred to residential PTSD programs when other less intensive treatment options have been exhausted. Thus, this population may have important differences from VA outpatient PTSD populations, as well as from veterans with different diagnostic pictures. Findings from this study should not be generalized to female veterans with PTSD, or male veterans with PTSD treated as outpatients or receiving care outside of the VA system, nor to nonveteran PTSD populations.

Although this sample was drawn from patients discharging from residential treatment, the average time from discharge to death (among those who patients who died during the study period) was roughly 4 years. This highlights the strong need for continuing care for veterans with chronic PTSD that allows for effective transitions to and from programs of various degrees of intensity (i.e., inpatient, outpatient, and community-based programs).

This initial study is limited by the relatively small sample size afforded by examining individuals from a single PTSD treatment program, and by variability in the number of years patients were followed from treatment to follow-up. However, this first study provides findings that are clinically important and suggest potential opportunities to enhance mortality and morbidity outcomes of this at-risk patient population through development of effective harm reduction interventions and improved continuity of care.

Acknowledgments

Preparation of this manuscript was supported in part by the Department of Veterans Affairs (VA) National Center for Posttraumatic Stress Disorder, VA Health Services Research and Development Service, and VA Palo Alto Health Care System. The authors acknowledge Jimmy Choi, Patricia Chuo, Michael D'Angelo, Betsy Egan, Gilbert Ramirez, and Bryan Robison for their efforts in obtaining data utilized in this study. The views expressed in this article are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs.

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